ORPHANHOOD AND HUMAN CAPITAL DESTRUCTION: IS THERE PERSISTENCE INTO ADULTHOOD?*

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This article presents unique evidence that orphanhood matters in the long run for health and education outcomes in a region of northwestern Tanzania. We study a sample of 718 non-orphaned children surveyed in 1991–1994 who were traced and reinterviewed as adults in 2004. A large proportion, 19%, lost one or more parents before age 15 in this period, allowing us to assess permanent health and education impacts of orphanhood. In the analysis, we control for a wide range of child and adult characteristics before orphanhood, as well as community fixed effects. We find that maternal orphanhood has a permanent adverse impact of 2 cm of final height attainment and one year of educational attainment. Expressing welfare in terms of consumption expenditure, the result is a gap of 8.5% compared with similar children whose mothers survived until at least their 15th birthday.

Childhood orphanhood is considered a major risk factor for poverty in adulthood through, among other channels, shortfalls in human capital investments in children. This article provides unique evidence on the long-term impact of orphanhood in Kagera, a region of Tanzania near Lake Victoria—an area ravaged by HIV/AIDS. The underlying data set is a 13-year panel data set in which individuals interviewed at baseline were traced irrespective of their current residence. This allows us to focus on non-orphaned children experiencing the loss of one or both parents during the survey period, controlling for their characteristics before becoming orphans. Furthermore, we can focus on the permanent impact in terms of height and educational attainment once these children reach adulthood and recovery is hardly possible anymore. Using within-sample estimates of the returns to height and education, we can also estimate the resulting lifetime welfare loss.

We find significant permanent education and health effects. Adults who had been maternally orphaned between the ages of 7 and 15 experienced a loss of, on average, nearly 2 cm of final attained height and one year of schooling. In contrast, paternal orphans have significantly lower height and years of schooling, but our analysis shows that a causal link does not seem to exist. Our projections also suggest that maternal orphanhood creates a lifelong deficit in consumption expenditure of roughly 8.5%.

Monasch and Boerma (2004) found that in sub-Saharan Africa, 9% of children have lost at least one parent, and one out of six households is caring for an orphan. The prevalence of orphanhood in the region has been greatly exacerbated by the HIV/AIDS pandemic. Because HIV in Africa is transmitted primarily through heterosexual contact, the epidemic is having a major effect on the mortality of men and women in their prime childbearing and earning years. With rising mortality rates and decreasing adult life expectancies, orphanhood rates in Africa continue to increase, and in hard-hit countries,

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AIDS is estimated to be a leading cause of orphanhood (UNAIDS 2004). In this article, we cannot identify the cause of deaths of adults, although HIV/AIDS is estimated to be a leading cause of death among adults aged 15–59 in Tanzania (CDC 2000). Kagera in particular was the worst-hit region in the country during the decade this study approximately spans (see the Data and Preliminary Analysis section). And indeed, in the data used in our study, as in other studies, the vast majority of prime-age mortality is attributed to disease or illness rather than to injuries or accidents. Our estimates constitute "mean" impacts across children orphaned by any cause. Nevertheless, these estimates still constitute the best—and arguably lower-bound—estimates currently available on long-term human capital outcomes for children orphaned through HIV/AIDS in Africa and, importantly, those orphaned in an environment with elevated mortality levels. Therefore, they are relevant to understanding the intergenerational impact of the HIV/AIDS epidemic.

Orphanhood is expected to influence health outcomes and schooling, although there are multiple potential pathways by which losing a parent may influence these outcomes. Obviously, the income effect is a strong candidate for a pathway, especially when parental deaths are associated with costs and income losses due to chronic illnesses. If households are credit constrained, then reduced incomes can result in lower investments in education. Aside from these direct wealth effects, orphanhood can be associated with an increased value of the child's time in home production (as a substitute for adult labor), which results in less schooling. There may be discrimination against orphans and favoritism toward biological children for double orphans or among single orphans who do not reside with their surviving parent (i.e., are fostered out). Beyond the financial consequences of adult deaths and the implications of a loss of parental involvement, children who become orphans may suffer trauma, which may, in turn, affect schooling and health outcomes. Children who lose a parent to AIDS specifically may be additionally stigmatized relative to other causes of mortality. In turn, lower investments in children (from orphanhood or other causes) can result in very large economic consequences, as demonstrated by Bell, Devarajan, and Gersbach (2006) in their simulations of the impact of orphanhood on economic outcomes in South Africa.

The available evidence of the impact of orphanhood typically relates to the impact of orphanhood from HIV/AIDS and other causes in the short run, often by examining a sample of school-age children in cross-sectional survey data. A small number of studies have used longitudinal data over short-run periods (perhaps 1–2 years) in which household coping strategies may successfully mitigate the impact. Studies of long-run impacts and outcomes are rare. Some of these studies are discussed in the next section. Understanding short-run outcomes is important, but short-run effects may not ultimately translate into worse welfare outcomes in the long run (that is, in adulthood). For instance, the socioeconomic consequences of a parent's death may affect outcomes around the time of illness or during a period of funeral/mourning. However, as extended families foster in orphans, these outcomes may recover over time.[1] Likewise, short-run coping might be unsustainable, and we might observe that households fall into a poverty trap as they deplete assets and other resources to maintain consumption.

In the next section, we review some of the existing empirical analyses. In the sections that follow, we discuss the data used in this study and then present the main empirical specifications, the results, and extensions. Conclusions are presented in the final section.

BACKGROUND

The literature on the consequences of orphanhood has tended to focus on education outcomes and use cross-sectional data, which may limit the robustness of the outcomes. The findings

1. See the studies cited in Golden (1994) regarding catch-up growth among stunted, malnourished children.

of these studies have been mixed,[2] as have the findings of the few studies looking at other outcomes, such as health. Few studies have also used panel data, which allow for extensive control of initial conditions.

Among the studies that used large, cross-sectional household survey data, several controlled for concurrent household characteristics to identify the impact of the loss of a parent on schooling. To the extent that orphans are found in relatively better-off households that also have higher demand for schooling, simple cross-sectional comparisons of enrollment rates between orphans and non-orphans may underestimate the true impact (see, e.g., Hargreaves and Glynn 2002; Ksoll 2007). On the other hand, depending on mortality patterns, in some settings, an orphan's household may be poorer prior to death, thus overestimating the impact of orphanhood.

Ainsworth and Filmer (2006) found that the orphan–non-orphan differential varied widely across the sample of countries they studied, which makes drawing conclusive statements difficult.[3] Case, Paxson, and Ableidinger (2004) used cross-section data; using household fixed effects, they found that orphans are disadvantaged relative to other children within the same household. However, it is not clear that a household fixed-effects approach is satisfactory. If orphans are strategically placed in better-off households within the extended family, then the orphans in a household fixed-effects framework are compared with a nonrandom sample of non-orphan coresidents. It is then not shown that orphanhood reduces schooling, but rather that orphans are placed with better-off relatives.[4] For instance, in their study of orphanhood in Zimbabwe, Nyamukapa and Gregson (2005) described the system of childcare arrangements as being traditionally based on both relatives' relationships with the orphan and their ability to assist. They further noted that orphan care arrangements are increasingly being influenced by financial considerations.

Evidence of other outcomes for orphans beyond schooling is much more scant, but here, too, the evidence is mixed (see, e.g., Chatterji et al. 2005). In one of the few studies that was able to identify actual cause of death of parents, Crampin et al. (2003) examined the health of children in Malawi linked with information on the HIV/AIDS status of parents measured 10 years prior.[5] They concluded that surviving children were not discriminated against as a result of parents having been ill or having died from HIV/AIDS. Another study of cross-sectional data (Lindblade et al. 2003) found that the health status of surviving orphans younger than 6 years is similar to their non-orphan counterparts in western Kenya.

2. Even estimates of the number of orphans is debated, with some research showing that the commonly cited statistics from the United Nations may seriously overestimate the number of orphans (Bennell 2005).

3. Even conclusions from the same data sets can differ depending on how data from countries are combined and which specific outcome is examined. Bicego, Rutstein, and Johnson (2003) combined Demographic and Health Survey household data across countries for West Africa and East Africa, whereas Ainsworth and Filmer (2006) did not merge data sets across countries. In other ways, the studies are very similar. Both examined school outcomes for orphans and non-orphans, controlling for sex, age, urban/rural residence, and household economic status. Additionally, Bicego et al. (2003) controlled for age, sex and education of the household head, and country; Ainsworth and Filmer (2006) also controlled for geographic region within country. Bicego et al. (2003) examined an indicator for the child being in the appropriate grade level, whereas Ainsworth and Filmer (2006) examined enrollment status. In West Africa, Bicego et al. found evidence of a significant impact among paternal orphans (6–14 years) and two-parent orphans (6–10 years). For East Africa, the findings were significant among maternal and two-parent orphans aged 11–14 years.

4. Gertler, Levine, and Ames (2004) addressed problems of nonrandom selection into orphanhood (i.e., the death of a parent being correlated with other disadvantages but not caused by the event of losing a parent) using propensity-score matching techniques. However, this matching is done between orphans and non-orphans based on characteristics of current residence, which may itself be endogenous. Information on residence prior to orphanhood may be more appropriate but was not available to Gertler et al.

5, Of the 2,250 offspring identified from the 593 individuals for whom the HIV/AIDS status at baseline was known, Crampin et al. were able to trace 1,141. Of these, 761 offspring were alive and interviewed, 167 were alive but left the district and were not traced, and 213 were deceased. It is unclear what the implications of this sample selection are on the measured impacts.

The studies cited above used cross-sectional data sets, which are limited to examining correlates of outcomes after a parental death, without controls for initial conditions prior to the death of the parent(s). These studies identified only who was an orphan and not how recently a parent died or the status of the child prior to being orphaned. Depending on mortality patterns of adults, there could be omitted variables that bias results.

Several recent studies have utilized available panel data to address these concerns. Using a panel survey from Tanzania covering 1991–1994 (the baseline for this study, described in the next section), Ainsworth, Beegle, and Koda (2005) found that adult deaths were associated with delayed enrollment among younger children (aged 7–10 years). Among orphans, younger maternal orphans were held back, whereas other orphans were not found to be disadvantaged. Case and Ardington (2006) studied schooling outcomes using a two- to three-year panel survey from South Africa. They found evidence of a causal effect of mother's deaths on children's education outcomes. Father's death, on the other hand, did not result in lower education, which instead was indicative of the socioeconomic conditions of the household prior to the death.

Ainsworth and Semali (2000) used the Kagera Health and Development Survey (KHDS) 1991–1994 data (the baseline for this study) to present random- and fixed-child-effects model estimates of adult death on children's height-for-age and weight-for-height. While random-effects results showed lower height for all maternal orphans and reduced height among paternal orphans in poor households, the fixed-effects results were not significant, possibly due to the small number of children who had any change in their orphan status between the survey rounds (about 21 months from the first to the last interview). They found no association between the loss of a parent (regardless of being coresident at the time) or recent adult death in the households and subsequent child weight-for-height. Overall, Ainsworth and Semali concluded that policy interventions should target poor households in general, among which the households hardest hit by adult mortality are likely to be found. They identified population-wide policy interventions—like universal availability of oral rehydration salts at health facilities, measles vaccinations, and improved physical access to medical care—as being most appropriate.

Using unique panel data from Kenya, Evans and Miguel (2007) studied a large sample of non-orphans enrolled in grades 1–7 in 1998 and reinterviewed in 2002. They evaluated the impact of orphanhood transitions on schooling participation (measured as the fraction of visits in the school year in which the child was in school on the day of an unannounced check). Maternal deaths lead to lower participation after the death, as well as in the one to two years before the death. Paternal orphans did not have lower school participation. It is unclear how the measure of school participation translates into completed school years, but presumably it implies lower overall attainment. They also examined the direction of omitted variable bias and concluded that unobservable characteristics of orphans led to underestimates of impact. Yamano and Jayne (2005) studied the impact of prime-age adult deaths on schooling for 7- to 14-year-olds using panel data from 2000 and 2002, which is similar, although not equivalent, to assessing the impact of orphanhood. They found much larger effects among children from poorer households. Girls suffered reduced schooling prior to the adult death, while boys experienced lower schooling after deaths.

Although existing studies are varied in their methods and conclusions, several themes emerge. Panel data analyses have shown that omitted variables (pre-orphan characteristics) can bias results. Further, there may be considerable heterogeneity across different types of orphans or in effects depending on the circumstances or characteristics of different orphans, such as gender. For example, orphans who are fostered from a young age may be more likely to be treated more like the children of the head if foster children, when fostered at young ages, display similar bonding with foster parents as biological children. Likewise, conditional on age, outcomes may differ depending on whether a child was already enrolled when s/he became orphaned. Bhargava (2005) analyzed a sample of orphans and found a

positive relationship between enrollment prior to becoming an orphan and subsequent enrollment rates among orphans. Lastly, since these studies focused on samples of children, their findings may not reflect the consequences in adulthood of the loss of a parent as a child.

This article contributes to the existing set of studies by presenting unique evidence of the extent to which orphanhood matters in the long run for health and education outcomes.[6] The data are from a region of northwestern Tanzania that is deeply affected by the HIV/AIDS epidemic. We use a sample of non-orphaned children aged 6–15 surveyed in 1991–1994 who were traced and reinterviewed as adults in 2004. Preliminary analysis in Beegle, De Weerdt, and Dercon (2006b) showed that a large proportion, 19%, lost one or both parents before the age of 15 in this period, allowing us to identify the permanent impact of orphanhood shocks. In the analysis, we can control for a wide range of child, parental, and household characteristics before the loss of the parent, as well as for community fixed effects.[7]

DATA AND PRELIMINARY ANALYSIS

The Kagera Region of Tanzania is located on the western shore of Lake Victoria, bordering Uganda to the north and Rwanda and Burundi to the west. The population (1.3 million in 1988, about 2 million in 2004) is overwhelmingly rural and primarily engaged in producing bananas and coffee in the northern districts and rainfed annual crops (maize, sorghum, and cotton) in the southern districts. The first three cases of HIV/AIDS in the Kagera region were detected at Ndolage Hospital in 1983.[8] Being one of the first regions in Africa to have been hit by the epidemic, it has a longer history of HIV/AIDS and therefore is one of the first places where one can attempt to analyze the intergenerational impact of the disease. Furthermore, Kagera had intensive socioeconomic data collection efforts early on in the epidemic, providing the necessary baseline data to perform such studies. In particular, this study uses baseline data from the Kagera Health and Development Survey (KHDS), a longitudinal socioeconomic survey conducted from September 1991 to January 1994 and covering the entire Kagera region (see World Bank [2004] and http://www.worldbank.org/lsms).

In 2004, another round of data collection was completed (Beegle, De Weerdt, and Dercon 2006a). The goal of the KHDS 2004 was to reinterview the sample of about 6,200 respondents from the 1991–1994 survey. Considerable effort was made to track surviving respondents to their current locations, be it in the same community (typically a village), a nearby community, within the region, or even outside the region or abroad. Excluding households in which all previous members were deceased (17 households with 31 people), the KHDS 2004 survey recontacted 93% of the baseline households (835 out of 895 households). The KHDS panel has an attrition rate that is much lower than that of other well-known panel surveys, which are summarized in Alderman et al. (2001). Notably, most of the surveys reviewed in Alderman et al. covered considerably shorter time periods (2–5 years). Refusals in the KHDS were relatively uncommon; the main reason for not reinterviewing surviving panel respondents was failure to locate the respondent.

The KHDS 2004 sample is remarkably similar to a random sample of households from Kagera, despite the fact that the baseline sample was not a simple random sample and given the attrition since the baseline. In the KHDS baseline, households with a sick or recently deceased adult were oversampled in enumeration areas; among the sample selected

6. This work also informs intergenerational models of the macroeconomic impact of AIDS, which take into account the impact on human capital formation and its transmission between generations. Both Bell et al. (2006) and Corrigan, Glomm, and Mendez (2005) made assumptions about the magnitude of the impact of the death of a parent without reference to any empirical studies of this link.

7. If parental mortality displayed high intracommunity correlation, then community fixed effects could remove much of the variation in mortality. We do not, however, find any evidence of this in the data in terms of clustering of deaths or deaths caused by localized epidemics.

8. Killewo et al. (1990), Killewo et al. (1993), and Kwesigabo et al. (2005) have provided epidemiological data on HIV/AIDS prevalence and incidence in the region.

at baseline, 5.8% did not participate, mainly due to the household having relocated after enumeration (53%). The households in the KHDS 2004 had characteristics not significantly different from those found in the Core Welfare Indicators Questionnaire (CWIQ) survey in 2004 (including characteristics such as household size, female headship, head's education, head's age, land holdings, livestock holdings, and dwelling characteristics). Among children under 18 years, 14% in the KHDS 2004 were single- or double-parent orphans, and 63% of children were living with both parents. These numbers are strikingly close to results from the Demographic and Health Survey (DHS) 2003. The DHS 2003 survey found that 11% of children under 18 years old in Kagera had lost one or both parents (a number equal to the rate in the Tanzanian national sample). In the DHS, 60% of children in Kagera who were under 18 years of age lived with both parents.

The sample of households includes both orphaned and non-orphaned children. In the latter group, at baseline, there were 1,085 non-orphaned children aged 6–15 for whom full baseline socioeconomic and anthropometric information is available and who would be at least 19 years old by 2004. Of these children, 822 (76%) were reinterviewed in 2004, a substantially lower recontact rate than the household measure reported above. Most of this attrition is due to the inability to locate movers (4% had died prior to 2004, and 20% had moved and were not located). Unfortunately, we do not have educational outcomes for these children, which could have potentially been reported by other household members, although perhaps not very reliably. Roughly half were traced outside of the baseline community, highlighting the importance of our tracking exercise to keep attrition rates low. In a supplemental appendix available on *Demography*'s Web site (Appendix A, at http://www.soc.duke.edu/resources/demography), we present a brief analysis of the characteristics of the individuals lost in the sample. Building on Becketti et al. (1988) and Fitzgerald, Gottschalk, and Moffitt (1998), we show that despite less successful rates of reinterview for children with some observable characteristics, a number of corrections for attrition show that the results are unaffected by this attrition. Of these 822 tracked children, we are missing information on 2004 schooling for 2 respondents, baseline school status for 7 respondents, and 2004 anthropometrics for 95 respondents. So we have complete socioeconomic and anthropometric data from baseline and follow-up for 718 children, which is an effective follow-up of 66%.

Even when parents are alive, children do not necessarily reside with them. As is the case in other sub-Saharan countries, the rate of fostering of children is high in Tanzania. While fostering is associated with orphanhood (i.e., two-parent orphans must be fostered and one-parent orphans are more likely to not live with a parent than children with two living parents), many non-orphaned children are not residing with one or both surviving parents.[9] In the KHDS, one-parent orphans are significantly less likely to be residing with the surviving parent than children with both parents alive. In the baseline, 86% of non-orphans were residing with at least one parent. Among one-parent orphans, however, 67% were residing with the surviving parent.

Table 1 shows that out of our sample of 718 children, who at baseline had both parents alive, 133 lost either one or both parents before the age of 15. The sample includes 52 people who lost their mothers and 98 people who lost their fathers before the age of 15. Only 17 people became double orphans before age 15.

Table 2 shows that non-orphaned children aged 6–15 at baseline who subsequently lose a parent will, by adulthood, be an average of 2 cm shorter and have one year less schooling than those who do not. Paternal and, especially, maternal orphanhood at childhood are

9. Despite the high rate of fostering among both orphans and non-orphans, some studies have used fostering as a proxy measure of orphanhood. For example, Deininger, Garcia, and Subbarao (2003) drew conclusions regarding the consequences of orphanhood using longitudinal household data from Uganda with information on child fostering. Yamano, Shimamura, and Sserunkuuma (2006) concluded that these results overestimate the negative impacts for orphans.

Table 1. Transitions in Orphan Status Among Non-orphans Aged 6–15 at Baseline

Variable	*N*	Percentage
Both Parents Alive at Age 15	585	81.4
One Parent Died Before Age 15	133	18.5
Mother Died Before Age 15	52	7.2
Father Died Before Age 15	98	13.6
Double Orphan by Age 15	17	2.3
Number of Observations	718	
Mean Age When Orphaned Among Orphans	11.3	

Notes: The sample consists of 718 children who were 6–15 years old and not orphaned at their baseline interview (September 1991–January 1994), and were subsequently reinterviewed in 2004. Because lost mother and lost father categories include double orphans, rows do not add up to 100%. For double orphans, the younger age is taken to calculate mean age orphaned.

Table 2. Average Adult Height and Schooling Attainment of Orphans and Non-orphans

Variable	Mean Final Height, Non-orphans (cm)	Mean Final Height, Orphans (cm)	Difference in Mean Final Height (cm)	*t* Statistic of Difference	*p* Value of Difference (one-sided)
Either Parent Died Before Age 15	162.83 (585)	160.93 (133)	1.90	2.47	.007
Mother Died Before Age 15	162.65 (666)	160.30 (52)	2.35	2.03	.021
Father Died Before Age 15	162.69 (620)	161.10 (98)	1.59	1.83	.034
Variable	Mean Final Schooling Non-orphans (years)	Mean Final Schooling Orphans (years)	Difference in Mean Final Schooling (years)	*t* Statistic of Difference	*p* Value of Difference (one-sided)
Either Parent Died Before Age 15	6.24 (585)	5.44 (133)	0.80	2.82	.003
Mother Died Before Age 15	6.13 (666)	5.62 (52)	0.52	1.21	.113
Father Died Before Age 15	6.22 (620)	5.33 (98)	0.89	2.77	.003

Notes: Numbers of observations are shown in parentheses. See the footnote in Table 1 for a description of the sample.

statistically significant predictors of lower adult height, while only paternal orphanhood is statistically associated with lower education in adulthood. These correlations, however, do not control for other socioeconomic differences in these two groups of children, differences that could partially or fully explain the height and education gaps observed.

Before modeling the link between human capital indicators and orphanhood, we look at the household characteristics and socioeconomic status during the baseline survey of those who became orphans relative to their non-orphan counterparts, using simple significance tests of the differences in the means. As shown in Table 3, those who will experience the

Table 3. Difference in Baseline Characteristics by Future Orphan Status

Baseline Characteristic	Remains Non-orphan to Age 15		Loses One or Both Parents Between Ages 6 and 15		Level of Significance
Child Height (cm)	133.64	(14.97)	126.61	(12.18)	†
Child Completed Years of Schooling	1.71	(2.06)	0.85	(1.43)	†
Age	10.90	(2.74)	9.56	(2.38)	†
Male[a]	0.48	(0.50)	0.41	(0.49)	ns
Mother Resides in Household[a]	0.78	(0.41)	0.65	(0.48)	†
Father Resides in Household[a]	0.78	(0.41)	0.64	(0.48)	†
Household Head Years of Schooling	4.48	(2.84)	3.55	(2.78)	†
Household Head Male[a]	0.90	(0.29)	0.79	(0.41)	†
Household Head Age	50.48	(13.43)	54.47	(13.75)	†
Ln per Capita Consumption (TZ shillings)	11.28	(2.39)	11.53	(1.84)	ns
Dwelling Has Good Flooring[a]	0.15	(0.35)	0.16	(0.37)	ns
Height of Mother (cm) Conditional on Residing in Household	157.65	(5.64)	158.16	(6.34)	ns
Number of Observations	585		133		

Notes: Standard deviations are in parentheses. See the footnote in Table 1 for a description of the sample. Level of significance is noted from a (two-sided) *t* test for the difference between the those who remain orphans and those who lose one or both parents between ages 6 and 15.

[a]Indicates binary (0/1) variables.

†$p < .10$; ns = not significant at $p < .10$

death of one or both parents before the age of 15, on average, are shorter and have less schooling. Among the set of other characteristics, future orphans are less likely to live with either their mother or father at baseline. The heads in the households in which they were living during the baseline were older, less educated, and more likely to be female. Further, they do not appear to be wealthier in terms of household consumption per capita or quality of flooring in the dwelling.[10] The height of their mothers is also not significantly different. This would suggest that socioeconomic differences determining selection into orphanhood are possibly not as strong as in some other studies.[11]

These descriptive statistics suggest two things. First, orphanhood itself may be a nonrandom event that is correlated with poorer health and education outcomes. That is, a simple difference-in-difference analysis may not show a negative impact of orphanhood on height and schooling because baseline differences exist. Second, even if height and schooling outcomes were not different when the children were young, other baseline (pre-orphanhood) characteristics might differ, such as household wealth and fostering propensities, which may influence long-run outcomes. In this case, one would want to carefully control for these baseline characteristics to make inferences about the impact of subsequent orphanhood. Still, unobservables correlated with orphanhood may bias results,

10. Household consumption expenditure is the aggregate of food and nonfood expenditures by households; it is measured based on the standards of the Living Standards Measurement Study survey program of the World Bank (see Deaton and Zaidi 2002). Food expenditure is the total of actual food purchases and the value of home consumption.

11. Using panel data, Case and Ardington (2006) found that paternal orphans had lower socioeconomic status. Their work concluded that the death of the father does not cause lower socioeconomic outcomes, but that poverty may have contributed to these deaths.

Table 4. Predicting Baseline Outcomes by Future Orphan Transitions

	Dependent Variable			
	Ln Height at Baseline		Years of Schooling at Baseline	
Variable	(1)	(2)	(3)	(4)
Mother Died	–0.007	–0.007	–0.150	–0.095
	(0.008)	(0.008)	(0.146)	(0.146)
Father Died	–0.010	–0.013	0.242	0.197
	(0.011)	(0.011)	(0.192)	(0.191)
Controls	Age, Sex	Full Set	Age, Sex	Full Set
Number of Observations	718	718	718	718

Notes: OLS estimates with community fixed effects. Standard errors are shown in parentheses. Full set of controls consists of child characteristics (sex and age dummy variables) and baseline characteristics (residing with mother and residing with father; years of education of mother and father; household consumption and flooring material; and age, years of education and sex of the household head). See the footnote in Table 1 for a description of the sample.

since even after observables are controlled for, orphanhood might not be random. Finding that orphanhood has a significant impact may not actually point to a causal impact, but rather may capture effects of unobservable covariates. For example, if particular attitudes toward health lead to parental illness and death as well as to poor health outcomes of the child, then orphanhood in itself is not the cause of lower height attainment in the child. An alternative narrative could also be that the *illness* of the parent really causes the health or educational deprivation of the child, and not the actual death of the parent.

We explore these possibilities by considering the relationship between future orphan status and baseline height and schooling with a minimal and an expanded set of control variables. We would expect that future orphan status would not be correlated with current outcomes unless (1) orphanhood captures some unobservable characteristics for which we are unable to control, or (2) morbidities associated with deaths affect outcomes before becoming an orphan. This approach follows Case and Ardington (2006), who made a causal interpretation of maternal orphanhood in a panel (2001 to 2003/2004) by showing no effect of future orphan status on current schooling. Likewise, Evans and Miguel (2007) noted the same issue in the context of censoring of the last round of their panel data, since they included post-orphan variables, which by definition are unknown for non-orphaned children in the final survey round.

Table 4 presents the regressions results of the correlates of height and years of education completed at the time of the baseline survey. The first pair of regressions for each outcome (columns 1 and 3) control only for age and gender. The second pair of regressions (columns 2 and 4) include the full set of baseline control variables, identical to those used in the next section (for long-run impacts). For both specifications, we find that future orphanhood is not correlated with baseline height or education, suggesting that unobservables correlated with health and education are not correlated with becoming an orphan in the future.

MODELING HEALTH AND EDUCATION OUTCOMES: METHOD AND RESULTS

We are interested in assessing the impact of orphanhood shocks on long-run health and education outcomes for non-orphans aged 6–15 at baseline. The basic specification for health in this evaluation can be written as

$$H_{i1} = \alpha + \beta \mathbf{X}_{i0} + \gamma D_i + \delta H_{i0} + \varepsilon_{i1}, \quad (1)$$

in which H_{i1} is health status in 2004; the vector $\mathbf{X}_{i0}$ is a set of individual, household, and community control variables measured at baseline, which is the first KHDS interview of the child in 1991–1994; and H_{i0} is the health status at baseline. The treatment of interest, D_i, is losing a parent between the baseline interview and the final interview in 2004. Thus, these deaths occur sometime between Period 0 (October 1991–January 1994) and Period 1 (2004). Estimated using ordinary least squares (OLS), Eq. (1) is equivalent to a difference-in-differences specification with a set of controls, including initial health, and it can be straightforwardly shown, by subtracting H_{i0} from both sides of Eq. (1), that the parameter γ offers the average treatment effect. Controlling for initial health status ensures that initial heterogeneity correlated to health endowments, health history, and general child background (as it affects changes in health status via H_{i0}) is captured.[12] The basic specification for education is identical, using educational attainment at baseline and in 2004 instead of the health status variables.

The outcomes used in this study relate to investments that affect long-term economic prospects of children. We focus on height as the key long-term health measure, since it has been shown to affect wage-earning capacity as well as participation in the labor force for men and women (see Haddad and Bouis 1991; and Thomas and Strauss 1997). We model height as influenced by several factors, including the characteristics of the child (sex and age, via a full set of age dummy variables), genetic background of the family (using height at baseline for both the child and the mother), and the socioeconomic environment in the household at baseline, which is assumed to influence human development investment (including whether the child was living with the mother, whether the child was living with the father, years of education, sex and age of the household head, and two indicators of wealth: cemented floor in dwelling and log per capita household consumption; see footnote 10).

Educational attainment can be used as a proxy for human capital levels. We focus on educational attainment in terms of the years of education completed, counting each grade completed as a year. We use a specification similar to the one above, whereby years of education in 2004 are determined by several factors, including child characteristics, years of education completed, and the same household characteristics as above. By controlling for years of education completed and whether enrolled in school at baseline, we further isolate the effect of orphanhood on education, net of its correlation with other unobserved background characteristics of these families influencing education. Finally, all our results control for community fixed effects at baseline, isolating the impact of orphanhood on health and education from factors such as access to schools and health services.

Baseline outcomes of height and schooling could be endogenous to children's subsequent growth and thus to their 2004 levels. We therefore opt to instrument these baseline outcomes, using two-stage least squares estimation (2SLS). We use as instruments shocks that occurred prior to the baseline and that can reasonably be expected to affect baseline outcomes but not subsequent growth (unless the effect is *through* baseline outcomes). Height and initial schooling at baseline are likely to be correlated with socioeconomic shocks during childhood. We use two variables for the instruments: past rainfall and more recent shocks. Past rainfall data were collected from the Kagera meteorological station, which maintains 22 rainfall stations in the region. Our rainfall variable is the deviation in millimeters from normal rainfall patterns when the child was 2–3 years old because events in early childhood have been shown to be a cause of stunting and low educational achievement (Glewwe, Jacoby, and King 2001). Past crop shocks are measured in the baseline survey with respect to shocks that impacted crop yields and farm harvest. These are likely to be valid instruments because their effects will be visible throughout childhood (Martorell 1999). Following Beegle, Deheija, and Gatti (2006), we also use the share of harvest lost in the year prior to the survey as an instrument.

12. Results are in logarithm of height and are consistent with using height in levels.

These regressions are then repeated with various different specifications to explore and assert their robustness, including the OLS difference-in-differences estimator (i.e., without instruments for baseline outcomes) and the same regression without including baseline height or schooling, simply looking at 2004 attainments. As a final robustness check, we balance treatment and comparison units in terms of the baseline characteristics that are likely to influence orphanhood. We trim our sample according to the parameters specified in Crump et. al. (2006), in which the propensity score s should be in the interval

$$\left(0.5 - \sqrt{0.25 - \gamma^{-1}}, 0.5 + \sqrt{0.25 - \gamma^{-1}}\right),$$

with γ the solution to

$$\gamma = 2E\left[\frac{1}{s(s-1)} \middle| \frac{1}{s(s-1)} < \gamma\right].$$

Following Hirano and Imbens (2001) and Hirano, Imbens, and Ridder (2003), we then present efficient estimators of the average treatment effect on the treated by running a weighted regression of the orphanhood dummy variable D_i on the 2004 outcome variable H_i, as in Eq. (2).

$$H_{i1} = \alpha + \gamma D_i + \varepsilon_{i1}. \tag{2}$$

Treatment observations are weighed at unity, while control observations get a weight of $s / (1 - s)$. Propensity scores are predicted from a probit model that includes all covariates used in the OLS regressions described above (including village dummy variables). Note that this setup allows us to enter only one treatment variable at a time. The coefficient γ is the average treatment effect on the treated.

Table 5 shows the results of the basic regressions, allowing for a separate effect of maternal and paternal orphanhood and with full controls for baseline and child characteristics and instrumented baseline outcomes. The reported coefficients are from the 2SLS estimator of the average treatment effect of maternal and paternal mortality, with endogenous initial height/schooling. Full first- and second-stage results as well as some instrumental variables (IV) diagnostics are given in supplementary tables (Appendix B) available on *Demography*'s Web site.[13] To make the results between health and education fully comparable, we use effectively identical specifications and samples for both outcomes. Table 6 shows the results of a number of alternative specifications of this model, as discussed above.

Based on Table 5, the effect of maternal orphanhood on height amounts to just over 1%, or 2 cm of height lost. Since the average age at which children in the sample became orphaned is 11 years, this implies that shocks impact growth in teen and preteen ages. This phenomenon, although to our knowledge never documented for developing countries, has been studied in developed countries—for example, in research on anorexia (Davis et al. 1978; Modan-Moses et al. 2003; Pugliese et al. 1983). The results on education show an impact of maternal orphanhood of more than one year. The educational loss of maternal orphanhood from ages 6–15 is significant and substantive: it does not just delay education but reduces it by more than one year by adulthood. This represents 42% of one standard deviation of years of education in this sample. Table 5 further shows that paternal orphanhood, while negatively correlated with final height and schooling in the bivariate statistics of Table 2, does not appear to have a causal impact.

13. The instruments are strongly significant in the first-stage regression and not "weak," provided that we allow for a size distortion of the IV estimates of around 25% of the bias using the critical values of Stock and Yogo (2005).

Table 5. Determinants of Height and Years of Schooling in 2004

	Dependent Variable	
Variable	Ln Height (1)	Years of Schooling (2)
Mother Died	–0.012†	–1.240*
	(0.006)	(0.423)
Father Died	–0.0003	–0.324
	(0.004)	(0.306)
Number of Observations	718	718

Notes: 2SLS estimates with community fixed effects. Standard errors are shown in parentheses. Includes controls for child characteristics (sex and age dummy variables) and baseline characteristics (residing with mother and residing with father; years of education of mother and father; household consumption and flooring material; and age, years of education and sex of the household head). Regression (1) includes the instrumented height of the child at baseline and, when available, mother's height from the baseline data. Regression (2) includes the instrumented number of years of education of the child at baseline. Instruments are, respectively, share of harvest lost in the year prior to baseline and rainfall deviation during long rains at age 2–3. *F* (Cragg-Donald) "weak" identification tests have the value of 5.58 and 4.95 respectively, suggesting an approximate size distortion in the IV estimates of about 25% of the OLS bias (and somewhat more for the schooling regression) (as in Stock and Yogo 2005: Table 2). The first-stage equation is exactly identified. See the footnote in Table 1 for a description of the sample.

†$p < .10$; *$p < .05$

Table 6 repeats these results for health with different specifications. The size and significance of the impact of maternal death on adult height and schooling remain robust to uninstrumented initial height and initial schooling (panel 1) and to excluding initial height and initial schooling (panel 2). Panel 3 presents an alternative estimator based on a trimmed sample and estimates average treatment effects through a weighted regression with unity weights for treatment observations and $s / (1 - s)$ weights for controls, where s is the estimated propensity score. Depending on the specification, samples with propensity score estimates below 0.04–0.08 and above 0.92–0.96 are excluded, following the criteria set out in Crump et al. (2006). Again, we find no impact of paternal death and a large and significant impact of maternal death on adult height and schooling.

These regressions control only for the gender of the child by using a dummy variable. Further analysis (not presented) showed that the impact of orphanhood had no gender dimension—the impact was never significantly sex-dependent. The potential additive effect of losing both parents was also tested and found not to be significant. This result should be treated with caution, however, because there are few double orphans in the sample. Double orphanhood also did not affect the magnitude of the effects of orphanhood episodes, so it is not considered further.

Unpacking the results further, we explore whether there is any heterogeneity in the impact of orphanhood in other dimensions. For example, we investigate whether the impact on health and education differs with family wealth by interacting the orphanhood variable with the baseline consumption expenditure variable. The interaction term is not significantly different from zero. Ideally, with a sufficient number of events, we would study these impacts by age at which children are orphaned, particularly for preschool-age children or at least those near to 6 years old when orphaned. However, we have few orphanhood events at very young ages to undertake this analysis.

Finally, we investigate whether there is any impact of different living arrangements. In principle, understanding the impact of fostering and other living arrangements as a coping

Table 6. Robustness Tests of Table 5 Results: Alternative Specifications

Variable	Dependent Variable: Ln Height (1)	Dependent Variable: Years of Schooling (2)
Panel 1: OLS Cluster FE Without IVs for Initial Schooling/Health		
Mother died	–0.012†	–1.181†
	(0.006)	(0.389)
Father died	0.000	–0.352
	(0.004)	(0.297)
Number of treatment observations, mother died	52	52
Number of treatment observations, father died	98	98
Number of observations	718	718
Panel 2: Cluster FE Without Initial Schooling/Health		
Mother died	–0.015*	–1.059†
	(0.006)	(0.406)
Father died	–0.001	–0.411
	(0.005)	(0.311)
Number of treatment observations, mother died	52	52
Number of treatment observations, father died	98	98
Number of observations	718	718
Panel 3: Propensity Score Weighted Regression		
Mother died	–0.019*	–1.134*
	(0.009)	(0.457)
Number of treatment observations	46	46
Number of observations	364	364
Father died	–0.007	–0.494
	(0.006)	(0.394)
Number of treatment observations	86	86
Number of observations	373	373

Notes: Regressions in Panels 1 and 2 are specified as those of Table 5, but with different specifications as documented in the panel's heading. Following Hirano et al. (2003), Panel 3 estimates average treatment effects through a weighted regression with unity weights for treatment observations and $s / (1 - s)$ weights for controls, where s is the estimated propensity score. Samples were trimmed following the criteria set out in Crump et al. (2006). Depending on the specification, observations with propensity scores below 0.04–0.08 and above 0.92–0.96 were dropped. Standard errors are in parentheses. See the footnote in Table 1 for a description of the sample.

†$p < .10$; *$p < .05$

mechanism after orphanhood is of considerable interest, not least for policy responses, but is not easily investigated because the placement of orphans may well be correlated with unobservable child and household characteristics, affecting inference on the heterogeneity of the impact of orphanhood in the data.[14] However, we can explore the consequences of living arrangements at the baseline, before orphanhood, by interacting the orphanhood shock with whether the child was living with the respective parent that subsequently died at the

14. For example, abler or healthier children may more easily be adopted by other families. Ksoll (2007) used a subsample of the KHDS data set to explore the placement of double orphans based on a further survey of the extended family network.

time of the baseline survey. As discussed in the second section of this article, a substantial number of our sample of the non-orphaned children at baseline were not living with their mothers or fathers in that period (Table 3). For height, we find no significantly different effect according to living arrangements, and for education, we find that the entire impact of maternal death on schooling is confined to those children living with their mother at baseline. Fostered children have no significantly negative schooling effect from orphanhood.[15] It may be that fostering can shelter children from the negative effects of orphanhood in the data, although we cannot identify the impact of fostering prior to orphanhood. Still, placement issues may also trouble this result—for example, if only more able children are taken out of households with ill parents to continue their education—so caution is warranted.

EXTENSION OF THE MAIN FINDINGS

Finally, what do these losses in terms of health and education mean for the overall loss in living standards brought on by orphanhood? Ideally, to quantify these consequences, we should estimate earnings functions to obtain the returns to height and education. However, we do not have income data within the data set. Instead, we can estimate returns using total consumption expenditure in 2004, providing a useful proxy for overall living standards based on the sample studied in this article, who were young adults in 2004. We first use a simple specification, consistent with the standard Mincerian earnings regression, in which consumption expenditure per capita is regressed on the logarithm of height and years of education, controlling for a full set of age dummy variables and sex. However, this is unlikely to correctly measure the marginal effect of height and education because factors such as health and education endowments and family background, which are likely to be correlated with height and education, are not included. We therefore include a second specification using all household- and parental-level baseline controls (including wealth variables, parental education, and mother's height) and community fixed effects (i.e., a full set of controls, as used in Table 5).

Table 7 reports the coefficients of interest of these regressions, finding very strongly significant effects from both health and education in both specifications. When background controls are not included, the bias in estimated effects is in the direction expected, although small; returns are lower when controls are included. A 1% height gain adds 2.1% to consumption per capita, while an extra year of education adds 4.8% to consumption per capita for this sample of young adults.[16] Based on our results in Table 5, this would imply an overall loss in consumption per capita of about 8.5% due to orphanhood.

CONCLUSIONS

With this article, we seek to inform on the impact of mortality through its effect on the human capital of future generations. Based on a sample of children first interviewed between 1991 and 1994 and then reinterviewed as adults in 2004, this article provides unique and robust evidence of the long-term effects of the loss of a parent on children's human capital build-up. The sample underlying this study is unique in that the project spent considerable resources tracing all individuals initially interviewed, even if they had started their own households or moved out of the village by 2004. Attrition rates would

15. In this case, we also find some significant effect of paternal death for those living with their father at baseline. The effect is considerably smaller than for maternal death for those living with their mother at baseline. At baseline, we observe no significant difference in schooling outcomes among fostered and nonfostered children in this sample.

16. To our knowledge, there are no comparable estimates of returns to health for Tanzania. Recent careful estimates on the returns to education in the period 1993 to 2001, based on employees in manufacturing enterprises, suggest a return for a cohort of workers below 30 years of age of about 9% per year (Söderbom et al. 2006). Because our sample includes agriculture and other sectors, the fact that our estimated returns are lower should not come as a surprise. Nevertheless, it also implies that our estimate of the cost of orphanhood may well be an underestimate.

Table 7. Effect of Attained Schooling and Height on 2004 Consumption per Capita (OLS)

	Dependent Variable	
Variable	Ln per Capita Consumption 2004 (1)	Ln per Capita Consumption 2004 (2)
Ln Height 2004	2.400**	2.118†
	(0.662)	(0.695)
Years of Schooling 2004	0.078**	0.048†
	(0.009)	(0.010)
Controls	Age, Sex	Full Set
Number of Observations	681	681

Notes: Standard errors are shown in parentheses. Full set of controls consists of child characteristics (sex and age dummy variables) and baseline characteristics (residing with mother and residing with father; years of education of mother and father; household consumption and flooring material; and age, years of education and sex of the household head). The sample includes both orphans and non-orphans.

†$p < .10$; **$p < .01$

have doubled if respondents had not been tracked outside of the village. Controlling for a wide range of household and child conditions before orphanhood and for community fixed effects, we find evidence of persistent and causal impacts of becoming a maternal orphan before the age of 15. Paternal orphanhood, while correlated with lower final height and years of schooling, is not found to have a causal link. The larger effect for maternal deaths is clearly a pattern emerging from the existing literature. We find that maternal orphanhood has a permanent adverse impact of 2 cm on final height and one year of schooling. Extending this to adult welfare in terms of consumption expenditure, maternal orphans suffer a reduction of 8.5% in consumption expenditure.

This first quantification of the long-run causal effects of orphanhood is an important reference point for policy makers for several reasons. First, the results plainly show that orphanhood is not a temporary problem, but that its negative effects persist into adulthood and will jeopardize the human capital of a whole generation of Africans. Second, the results place serious doubts on the common practice of orphan assistance programs to enroll only those children who have lost their fathers. This article convincingly shows that even in societies where fathers are expected to be the main income earners, maternal death is important. Third, our analysis tries to address causality. Establishing causality is key to informing policy. Irrespective of any correlation between parental death and human capital outcomes, if there is no causal link, then tackling mortality would not be expected to contribute to increases in human capital investments of future generations. In fact, in this scenario, they constitute competing budget aims. Perhaps the most controversial conclusion of our study then is that while reductions in maternal mortality have a causal positive impact on the human capital outcomes of children, reductions in paternal mortality do not. In terms of budget allocations, this means that resources spent on reducing maternal mortality are complementary to the goal of increasing human capital outcomes of future generations, while resources spent on reducing paternal mortality may compete with this goal. This does not mean that reducing paternal mortality is not important in other dimensions of welfare, and neither does it contradict our corollary finding that paternal mortality is an important indicator identifying vulnerable children.

Specific policy prescriptions from this work and other such studies remain elusive because multiple potential policy responses exist. This article cannot address whether, for example, one should offer antiretroviral therapy to AIDS-infected women, reduce other

forms of maternal mortality, scale up HIV/AIDS prevention campaigns, or provide scholarships to maternal orphans. In this sense, the findings here are interesting in general but perhaps more so when the context is considered. In parts of this region of Tanzania, substantial interventions for some households and villages have taken place, although still not at a sufficient scale to make any meaningful inferences from the small number of relevant observations in our sample. It is not possible to state from this study that the impact is at all affected positively or negatively by existing interventions. A randomized experiment would need to be set up for this—something that would raise obvious ethical concerns and would not yield results on long-run impacts until more than a decade from now. If anything, the impact exists despite any existing interventions and programs.

With these considerations in mind, in future work, the impact of specific programs and interventions will have to be explored further, as well as other long-term impacts of orphanhood and other shocks, such as the extent to which orphanhood is the main source of deprivation among children or just one of many factors relevant for targeting the poor and vulnerable. Establishing the size of the long-term impact of orphanhood on children, as achieved in this article, is nevertheless an essential first step.

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